MEDICAL ASPECTS OF VISCEROPTOSIS.

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NO RELIABLE INFERENCES REGARDING THE FUNCTION OF THE STOMACH CAN BE DRAWN FROM ITS POSITION IN THE ABDOMINAL CAVITY.

By gastro-enterologists it is universally conceded that even most pronounced degrees of ptosis may coexist with perfectly normal function.

Stockton says that gastroptosis is usually unaccompanied by symptoms sufficiently specific to lead one to suspect its presence. R. H. Smith says that "many enteroptotic women, even when the prolapse is great, have good digestion and are free from symptoms." Otto Strauss1 says that, in contrast to the atonic stomach, the prolapsed stomach often shows very good tonus; in the majority of cases it exhibits well-developed peristalsis, and very often empties itself in the same time as a normal stomach. Similar quotations could be cited in an unlimited number.

Conceding that many prolapsed stomachs functionate normally, what can we say concerning the many prolapsed stomachs which functionate badly? Let us hear what an X-ray specialist has to say on this subject. In an article on the "X-ray Diagnosis of Gastroptosis," H. K. Pancoast declares as follows: "Knowing that an apparent ptosis may exist without symptoms, given a case of suspected gastroptosis, the roentgenologist first determines whether the typical roentgen picture is present; he then observes the extent of the ptosis, the degree of atony and the delay in the time of clearance. Next he must be assured that the position of the stomach is not due to extra-gastric causes. Then he determines whether the atony and dilatation and the other factors in retention are those truly associated with a gastroptosis, or arise from other causes. Finally, it is important to examine the intestinal tract, because of the possible factors to be found in them." (This quotation is curtailed and paraphrased, but substantially accurate.) Now, such an exposition is little less than a reductio ad absurdum. We might as well say that if no other causes produce symptoms in a prolapsed stomach, possibly the prolapse itself produces them. In other words, the position of the stomach "per se" teaches us nothing regarding its function.
Roentgenologists have known for a long time that in the same individual the transverse colon can assume different forms and positions at different times. J. T. Case informs us that within five or six hours the transverse colon may assume half a dozen different shapes; that the level reached by the lowest border of the transverse colon varies considerably in different patients, as well as at different times in the same patient. In tall, slender individuals, the lower border of the transverse colon normally may reach several inches below the line joining the iliac crests, although in the same type of individual the transverse colon may lie well above the navel when the patient lies supine. He says further that "stasis in a prolapsed transverse colon is practically never encountered," and that he does not seriously consider ptosis of the transverse colon as a cause of constipation. Hopmann says that in many cases of prolapsed abdominal viscera the evacuations are perfectly normal.

No competent internist or roentgenologist any longer accepts the crude notion that prolapse of the colon produces kinks along its course, and that these kinks form obstructions and produce stasis. In the first place the apparent kinking in the roentgenogram is obviously an illusion due to one plane photography, and this illusion is easily dispelled by stereoscopic pictures. Keith calls attention to the fact that there is no hypertrophy of the muscular coats above the site of so-called bands or kinks, and that when acute flexures are produced experimentally (Murphy and Cannon) stasis does not result. Case says there is rarely any real kinking in the colon, even when adhesions are numerous.

A movable cecum (which is often discussed in connection with coloposis) is considered by many competent authorities, both medical and surgical, to be a normal condition.

Finally, when we consider the normal physiological mechanism by which the contents of the colon are pushed forward; the powerful colonic contractions, which in the course of a few seconds propel large columns of colonic contents onward a distance of six to twelve inches, or even further; how the column moves up hill and down dale with equal speed and facility, i.e., up the ascending loop of the transverse colon and around the splenic flexure; when we recall, furthermore, that the colon has muscular fibres calculated to be as voluminous as in the biceps of a blacksmith's arm, we must conclude that variations in position of and by themselves can have little, if any, effect on the normal function of the colon.
THE POSITION OF THE RIGHT KIDNEY IS OF IMPORTANCE ONLY WHEN IT PRODUCES LOCAL SYMPTOMS.

Moderate displacements of the right kidney are no longer considered pathological (Israel, Litten, Kuttner). Even patients with marked nephroptosis may have no symptoms. So much harm has been done to patients in the past by riveting their attention on the position of the kidneys, that all clinicians (both internists and surgeons) in recent writings warn us against communicating our knowledge concerning the position of their kidneys to our patients unless definite local symptoms are present, such as colicky or dragging pains, hematuria or Dietl's crises. Forchheimer⁷ says that many a patient has been made an invalid by failure to heed this precaution. Sailer thinks, in fact, that the only definite symptom surely referable to nephroptosis is the Dietl crisis.⁸

THE CLASSIFICATION OF CASES OF VISCEROPTOSIS.

Bearing the above facts in mind, I believe it is fundamentally wrong to classify the cases of visceroptosis clinically, according to the static conditions presented, instead of according to their symptomatology. A useful clinical classification should divide cases of visceroptosis into three groups.

Group 1. One or more organs are prolapsed, but the individual is in good health.

Group 2. The individual is sick and has prolapse of one or more organs, but can be relieved without reference to the ptosis.

Group 3. The patient has symptoms which cannot be relieved without special attention being given to the displacement of the abdominal organs and to the conditions which underlie and occasion them.

Basing my conclusions upon clinical experience over a long period of years, I should say unhesitatingly that the third group, which should form the real and only basis for our discussion, is by far the smallest of the three groups. Failure to recognize this fact renders much of the current literature on visceroptosis, not only futile, but even harmful. To consider visceroptosis a clinical entity of and by itself is to mislead the inexperienced clinician; is to arm the roentgenologist with a facile instrument of often unintentional deception; is to tempt the ambitious surgeon into fields of action in which he can do much harm, and is to lead even many experienced clinicians far afield into ill-considered generalizations and lines of action.
It should be emphasized over and over again that static conditions as such can never be a criterion of disease; that our health depends not on anatomical relationships, but on function; and that he would be a bold clinician, indeed, who would venture to make emphatic deductions concerning the functions of an abdominal organ, from its shape and position alone.

The usual custom of separating cases of visceroptosis into two groups, the congenital and the acquired, has many features of practical utility.

The word congenital, of course, is in one sense a misnomer. The ptosis is not inherited, but merely the predisposition thereto. Neither the stomach, the colon nor the kidney is actually prolapsed before the age of puberty. At that period, according to R. H. Smith, there is a widening of the pelvis and a compensatory narrowing of the waist. In the thin, relaxed and badly nourished child these changes are pronounced, and prolapse of the kidney and the pyloric end of the stomach occur, along with other changes. Smith says that the state of nutrition during adolescence, more than any other cause, influences the size and form of the chest and upper abdomen and that, once established, these do not change materially during life. The congenital type presents many other stigmata, which will be referred to later.

Again quoting Smith, we can describe the acquired type as it occurs in women who during childhood and adolescence were well nourished, sturdy of form, firm of tissue, and who had deep chests, capacious upper abdomens and retentive abdominal walls. Through the weakening effects of child-bearing, fatigue, overwork, or other physical or mental strain, these women may acquire more or less relaxation of tissues, a changed configuration of the body, and a certain degree of visceral prolapse. As a rule, the degree of prolapse is less in this type than in the congenital, but the symptoms may be quite as distressing. While the pronounced types of the congenital and acquired groups can be readily distinguished, there are naturally many mixed types which it is impossible to classify. In general terms we can say that the acquired types can usually be markedly benefited by hygienic and other measures, but that members of the congenital group can usually not be radically and permanently improved.

THE SO-CALLED CASES OF CONGENITAL VISEROPTOSIS SHOULD NOT BE CLASSIFIED AS CASES OF VISEROPTOSIS AT ALL.

It is an interesting commentary on our methods of thinking to note that, whereas congenital viseroptosis was long ago recognized as only
a part, and not always an essential or even an important part, of a condition of general constitutional asthenia, we have, nevertheless, retained the term visceroptosis as a designation for the entire condition. In 1899, Stiller invented the term, "asthenia universalis congenita," to describe what we still call congenital visceroptosis, and the same year H. Strauss spoke of this condition as a coördinated expression of the constitutional inferiority, "minderwertigkeit," of various organs. The term "habitus asthenicus," or constitutional asthenia, has since then also become prevalent.

The essential truthfulness of Stiller's presentation, as applied to a certain large group of cases, is generally accepted. I shall not take up your time by going over this well-known ground. Stiller laid especial stress on the long, narrow, flat thorax, the small bones, the slight panniculus adiposus, the mobile tenth rib, and what he called a vulnerable nervous system. His general conclusion has met with practically universal acceptance, viz: that the symptoms in this type of visceroptosis are not due so much to the visceral displacement as to the vitiated muscular and nervous system of the individual.

Intensive study of the "habitus asthenicus" has disclosed other constituent elements. Among the congenital defects of development are: failure of the colon to rotate completely into the right flank; failure of complete fusion between the right meso-colon and the posterior parietal peritoneum, resulting in cecum mobile (Wilms); failure of the layers of the great omentum to fuse. Goldthwait lays emphasis on the smallness of the spine, and the deformity of the lumbar vertebrae. He also accepts, as quite characteristic for this type, an abnormal shortness of the large and small intestines. He calls attention to the undersized heart, the small lungs, the slender feet with their unnaturally high arches. Other writers have noted that in this type the female genitalia are often poorly developed.

To the study of structure has been added the study of function. It has been found that in children of this build orthostatic albuminuria is not uncommon; weak digestion and constipation are prevalent, and Uhlman has recently demonstrated that the liver in these subjects is physiologically inferior (as determined by the ready appearance of galactosuria after the administration of 30 g. galactose.

As many of these patients show a lessened reaction to pilocarpin, i.e., a certain grade of sympatheticotonia, it is possible that the lessened hepatic function indicates a vitiated nervous system.

When we sum up these observations we find that we have gathered into one group certain individuals of a particular body-form or habitus.
who are apt to present some or many of the following characteristics: a vulnerable nervous system of neurasthenic type; a weak muscular system; certain skeletal defects; physiologically weak heart, kidneys, liver and digestive organs; displacement of one or more abdominal viscera. Chiefly through custom, we still refer to these patients as being "cases of visceroptosis," although the malposition of the abdominal viscera is only one item out of many; is, in fact, not always present; frequently does not play an important part in the symptomatology, and may easily become a misleading factor in the treatment if an undue amount of attention is paid to it. The error is commonly made of ascribing off-hand any existing digestive disorders to the ptosis as such; especially to assume that the constipation is the obvious result of the prolapse (although we know that prolapse of and by itself does not produce constipation), and to direct all our therapeutic efforts to changing the position of the viscera by bandages, rest cures, and finally by operative procedures. It would be a great advance if we dropped the term visceroptosis or splanchnoptosis when referring to these cases. The designation, habitus asthenicus, is preferable and not misleading. With still more reluctance should we be willing to speak of these patients as "macrosecles" or "hyperontomorphs."

As a matter of practice we are usually led into error when we treat our patients not as individuals, but as members of a group. Nothing is so easy as the making of sweeping generalizations, and nothing is more misleading. It may often prove useful to recognize the fact that our patient belongs to a general class, but to speak of him, without first dissecting him, as a carnivorous or an herbivorous type, to posit for him certain potentials of disease, to try for the good of the race to eradicate him, is in no sense a progress in scientific medicine, but rather a reversion to the era of Galenic dyserasiae,—before we knew that streptococci and not humors caused gall-bladder infection and tonsillitis. No one can read critically the anatomic studies underlying the classification of human beings into herbivora, hyperontomorphs and meso-ontomorphs without realizing what leaps the imagination is compelled to make and what violence must be done to careful and individual observation.

THE ACQUIRED FORM OF VISCEROPTOSIS IS THE ONLY CLINICAL FORM OF VISCEROPTOSIS AS SUCH.

Visceroptosis is acquired in many ways. The first causes to be accurately studied were those involving some damage to the supporting structures of the abdominal viscera. Whatever theories we hold regard-
ing the manner in which the abdominal organs are held in position normally, we all recognize the fact that in order to maintain this position there must be a state of equilibrium between the volume of the abdominal cavity and its contents. This equilibrium can be destroyed by increasing the volume of the cavity (as in laxity of the abdominal walls, and rupture of the perineum) or by diminishing the volume of the contents, as in wasting diseases, malnutrition (Glenard). Both factors may work together. What rôle is played by the suspensory ligaments of the organs and the so-called intra-abdominal pressure is still problematic.

Wiedkopf has very graphically described the development and symptomatology of visceroptosis when acquired as the result of weakening of the anterior abdominal walls. Thus repeated pregnancies weaken the walls; the abdominal cavity becomes too large for its contents; the small intestine sinks; the stomach, transverse colon and right kidney follow. Now for the first time these organs actually become suspended from their supporting ligaments and drag on them. Eating causes increase of the drag, and so does overfilling of the colon (constipation). The quantity of the food taken (Rovsing) is of more importance than its quality. Lying down lessens the drag and the symptoms. The lower abdomen bulges; the upper abdomen is retracted. Wearing an abdominal support gives relief. All this is simple and obvious enough and a matter of daily observation.

The orthopedic surgeons, under the lead of Goldthwait, have shown us the frequent dependence of visceroptosis on bad posture, i.e., a relaxed position of the trunk. Bad posture depresses the position of the diaphragm, relaxes the abdominal walls and forces the abdominal contents downward and forward. Habitual bad posture tends to render the ptosis chronic and to cause other structural changes which I shall not repeat here.

The question which naturally arises is, What causes the bad posture? The answer is equally obvious. Bad nutrition, sickness, overwork, overstrain, a mal-adjusted psychology, discouragement. These cause the letting-go of the voluntary muscles, the tonic state of involuntary muscles. The downward drag and the unnatural pressure in turn produce effects on distant structures; the patients develop lumbago, sciatica, villous arthritis of the knee. One would think the process would end there. The health becomes depraved, the organs functionate badly and pain occurs in many areas. But no! In the visceroptotic individual chronic disease of the kidney, arteriosclerosis, diabetes, constant high blood pressure, sclerosis of the liver, gallstones and acid indigestion
are frequently seen. In 1914 Goldthwait\(^14\) hesitated to suggest that the above mechanical conditions could lead to these diseases (a natural hesitation); yet in the Shattuck Lecture for 1915 he suggests that the position of the spleen may induce the profound anemias (p. 52); a kink in the bowel may cause an eye infection (p. 54); a glycosuria be caused by mechanical pressure on the pancreas (p. 51); epilepsy be the result of enteroptosis. On page 52 we are told that the pelvic organs cannot work rightly if the loose abdominal organs are crowded into the lower abdomen and pelvis, and on page 54 we learn that in the carnivorous type (the type in which the most pronounced ptoses occur) conception occurs easily and large families are common. No! a mechanistic conception of disease is helpful only so far as it clings to facts which can be established by observation. It becomes fantastic and leads far afield when it outstrips fact and gives rein to imagination. After all is said and done, the patient is always an individual with individual problems to solve. To call him names does not help him. If the abdominal muscles are weak, if a prolapsed stomach functionates badly, the fact must be determined by observation.

If a woman's torn perineum causes dyschezia and the constipation leads to headache and nervous disturbances, these facts must be determined for her on the merits of her own case. Are your patient's symptoms the result of anatomical abnormalities, flat-foot, coloptosis, relaxed posture, or are they due to fatigue, unhappiness, strain, under-nourishment? The discovery of anatomical abnormalities does not answer the question; it only propounds it more sharply.

"Jede Menschensorge ist Individual-sorge," said Pestalozzi. It is well to remember that every patient's problems are his very own.

**References.**

5. **Keith**: Lancet, August, 1915.
7. **Forchheimer**: Therapeutics of Internal Diseases, Vol. iv, p. 2.
12. **Arch. f. Verdi.**: November, 1915.